CHICAGO NEUROLOGICAL SOCIETY.

Joint Meeting with the Chicago Medical Society, Dec. 10, 1902.

The President, Dr. Wm. A. Evans, in the chair.

The Changes in the Spinal Cord and Medulla in Pernicious Anemia.—This was the title of an address delivered by Dr. Frank Billings.

Causes.—Among other things, Dr. Billings said that our knowledge of the cause of pernicious anemia is not satisfactory. We recognize certain contributory factors and infer a condition of toxemia which has not been proved. The disease so frequently follows the infections, like syphilis, malaria, typhoid, la grippe, *etc.*, that it is impossible to ascribe any specific relation to them. The disease occurs more frequently in women than in men. It may occur at any age, but it is more common in the fourth decade than at any other time in life. Intestinal parasites have been found present in the disease, especially the *Bothriocephalus latus* and the *Ankylostoma duodenale*.

The condition of profound anemia, with deformed red cells, the presence of fetal red cells in the circulating blood, the presence of an abnormal amount of iron in the liver, together with degenerative changes in the muscles and in the marrow of bone, is a reasonable hypothesis for the assumption of the presence of a circulating poison—a hemolytic toxin—as the fundamental cause of the disease. The source of the poison has been the subject of many theories.

It is not improbable from the evidence we have that pernicious anemia is due to some hemolytic toxin, but whether of bacterial or autogenetic origin we cannot at the present time definitely say.

Symptoms.—The symptoms of pernicious anemia are chiefly those due to a profound anemia manifested by weakness, lessened endurance, with dizziness, dyspnea, palpitation of the heart, *etc.* In probably a large percentage of the cases the body weight is preserved. There is a tendency to subcutaneous, submucous, retinal and other hemorrhages. Gastro-intestinal disturbance is very commonly present, and especially diarrhea. The appetite is often poor or capricious. The disease is associated with nervous phenomena in the great majority of cases; usually, however, subjective in character. The spinal cord lesions, which are now recognized as occurring in a small percentage of the cases, may appear as one of the earliest manifestations of the disease, or they may occur late, and may be manifested slightly or not at all up to the time of death.

Dr. Billings gave an analysis of forty-one cases of pernicious anemia that he had seen during the last few years. The consensus of opinion is that the nervous lesions are due to a toxic agent, which is also responsible for the anemia when it is present. The fact that the lesion does not involve, as a rule, a whole neurone or system; that the trophic cell is usually not disturbed in either the cortex, the spinal gray matter or the posterior ganglion, and that the ultimate primary change is one of degeneration of the nerve fiber itself, speaks for the effect of some blood-circulating toxin. The fact, too, that the brunt of the lesion occurs in that part of the tracts involved farthest removed from the trophic center, where the nerve fiber is the least protected by its nutritive cell, is evidence of a toxic cause.

Conclusions.—The conclusions which one may draw from a study of the subject are: (1) That there is a well-established relation of diffuse cord degeneration with pernicious anemia; (2) It seems probable that the hemolysis and the cord changes are due to the same toxin; (3) While the source of the toxin is unknown, the fact that gastro-intestinal disturbance is so common in the disease would lead one to suppose that it is of intestinal origin; (4) The diffuse degenerations of the spinal cord which occur in conditions without pernicious anemia do not appear to differ essentially from those of pernicious anemia; (5) It is possible that a common blood-circulating poison exists which may expend its force on the blood in one individual, on the nervous apparatus in another, and coincidently on the blood and spinal cord in others.

Dr. Sydney Kuh said that Dr. Billings, in speaking of the etiology of pernicious anemia, quoted the statistics of some foreign authorities based upon 270 cases of pernicious anemia. In these statistics the statement was made that of this number of patients 22.4 per cent. had suffered from syphilis, and from this the conclusion was drawn that syphilis is a factor in the etiology of pernicious anemia, although not a very important one. This statement Dr. Kuh believed was misleading. Erb and Kuhn, and he himself, had tried to find how frequent syphilis is among patients in general, and it is quite remarkable to see how exactly the statistics of all three agree, the conclusion being that 22 and 23 per cent. of patients have been infected with syphilis.

There is no doubt that the toxin theory of the origin of pernicious anemia is by far the most plausible one. A variety of diseases, as atheroma, pellagra, *etc.*, can produce lesions in the spinal cord exactly similar to those mentioned by Dr. Billings. Dr. Kuh had seen one case in which the changes in degree and extent corresponded very closely to what had been described and demonstrated by Dr. Billings. In this patient, who had syphilis of the spinal cord, the parenchymatous and interstitial changes seemed absolutely independent of the distinctly syphilitic changes, and he attributed them to the syphilitic toxins.

Dr. Maximilian Herzog thought that it was not absolutely necessary to attribute the changes in the cord to toxins, because secondary anemia never leads to such changes. Secondary anemias are never as severe as initial or primary anemias. Secondary anemias do not kill the patient; he dies from tuberculosis or from some other condition, but not from the anemia itself. Dr. Herzog had never seen a secondary anemia like a primary anemia, with a blood count of 300,000. He thought malnutrition was more responsible for the changes in the cord than the toxins.

Dr. E. R. Moras stated that from observation and reading, and from cases he had seen, he had come to the conclusion that the fundamental cause of pernicious anemia is a chemic poison which exists in a relative quantity in the blood of probably every human individual or animal. The absolute quantity in each individual is determined by the habits, environment and special occupation and diet. Chemical analysis demonstrates that the acid produced in those conditions which are analogous to those found in the blood and tissues of pernicious anemic patients, is formic acid.

Dr. Archibald Church stated that about nine months ago he had under observation five cases of pernicious anemia with nervous manifestations. He had notes of about eighteen cases. Three years ago he called attention to the fact that in some instances spastic conditions were present, and that these very frequently terminated in flaccid paralysis. As the disease progresses it takes on the type of an ascending myelitis. Paresthesia is sometimes followed by anesthesia. In at least two cases he had seen a band of paresthesia or hyperesthesia followed by anesthesia gradually approach the trunk and chest, reach the neck, and terminate in Landry's paralysis. He was convinced that in some instances the gray matter is seriously involved. Recently he had followed to a fatal termination a case in which the lower limbs were at first in a spastic condition, and subsequently became flaccid and finally paralyzed; bed-sores developed, and the muscles of the trunk showed degeneration. Later the muscles of the hand and forearm showed decided atrophy, with fibrillary degeneration.

A point of some importance in the clinical onset of this disorder is that the mental picture is somewhat peculiar, owing to the probable impoverishment of the nutrition of the brain. These patients often present eccentricities of character and temper; they have hallucinations of sight; very commonly they will misinterpret things; they see objects crawling over the wall or bed or in the air, and will describe them. Other symptoms were mentioned by the speaker. Cases with which he had come in contact have simulated three conditions, locomotor ataxia, multiple neuritis and myelitis.

The speaker spoke of the beneficial results following the use of injections of salt solution. Dr. E. F. Wells had told him of a case which he believed to be practically cured by the use of large injections of salt solution.

Dr. Billings, in closing the discussion, said his belief is that a *Bothriocephalus latus* which will attack a red blood cell will not attack a tissue cell, but at present our knowledge of the blood conditions in these cases is in its infancy.

The clinical cases related by Dr. Church he had also seen, and in the original address which he delivered in Boston he added four other cases of the disease. He had not been able to procure a necropsy in the cases in which death occurred, but he gave their clinical histories. Dr. Church and he were associated in at least one of these cases, and he observed it through most of its clinical course—the one he referred to as terminating like an ascending myelitis. In addition, the patient presented in her first two years of illness a secondary anemia as marked as any he ever saw.

He had tried all sorts of treatment, and from the first had used the one suggested by Dr. Church, and while in some instances it had been of benefit, it was his experience that the patient dies, in spite of the normal salt solution in any amount injected into the cord. The only good influence it has is that it fills up the blood vessels. He did not think the portal circulation is improved very much, or the intestinal tract cleansed very much by washing out four or five feet of the colon and leaving the rest of the small intestine untouched.